the ascending colon. No exploratory laparotomy should be completed without a careful search for abnormal conditions affecting this portion of the intestinal tract.

THE COMMONER TYPES OF GOITER—CLINICAL AND PATHOLOGICAL CLASSIFICATION*

WITH NOTES ON PREVENTION AND TREATMENT

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INTRODUCTION

THE recognition of a goiter-bearing patient is not always easy, nor is the classification of that goiter always so simple. Clinicians and surgeons as a whole often fail to distinguish exophthalmic goiter from adenomatous goiter with hyperthyroidism (toxic adenoma) and the relationship of the adenomatous goiter with hyperthyroidism to the adenomatous goiter without hyperthyroidism is not sufficiently emphasized even today in the non-goitrous districts. The proper treatment of thyroid disease depends upon the recognition of the type of goiter in question. Failure to recognize that there are several kinds of goiter has resulted in much harm and an occasional unnecessary death.

While it should be emphasized that the recognition and classification of goiters must, in the last analysis, be from clinical signs and symptoms backed up by careful basal metabolic rate tests; nevertheless there is a definite gross and microscopic pathological picture which varies with the different types of thyroid disease. The different histopathologic picture may be the tissue explanation of the different chemistry of toxic adenomata and exophthalmic cases as shown by the different response to Lugol's solution.

The object of this paper is to call attention to a simple, workable, clinical classification of diseases of the thyroid gland correlating the pathology of the removed glands with the different clinical types insofar as that is possible, and to set down a few short notes on diagnosis, prevention, and treatment of the commoner types of goiter.

HISTORY

Mobius [1] in 1887 suggested that the clinical symptoms of Basedow's disease were due to an abnormally increased activity of the thyroid gland. Greenfield [2] in 1893 was the first to demonstrate this relationship by showing hypertrophy and hyperplasia in the thyroid gland of six typical cases of what is now called exophthalmic goiter. In 1910 Kocher [3] practically differentiated true Graves' disease and adenomatous goiter with hyperthyroidism when he collected and described a large group of patients showing different reactions to iodin administration. He failed, however, to separate the groups clinically. It is interesting to note that Aschoff,[4] independently of Kocher and Plummer, also made a distinction between these diseases from the pathologic study of excised goiters. Since 1911 Plummer [5] has repeatedly emphasized and maintained that there were two separate and distinct types of hyperthyroidism, each associated with a distinctive pathologic change in the thyroid gland. In 1913 Doctor Wilson [6] reported that in exophthalmic goiter cases as described and diagnosed by Plummer, the thyroid always showed histologically parenchymatous hypertrophy and hyperplasia. Plummer's clinical observations were corroborated by Kendall's [7] chemical studies on thyroxin. Dubois [8] in 1916 published extensive observations on the basal metabolism rate in goiter patients, and Boothby [9] showed the indirect gasometer method of calorimity in estimating metabolism, a practical help in the diagnosis and treatment of thyroid diseases.

The two most important and recent advances in the handling of thyroid disease have been the use of iodin in the treatment of exophthalmic goiter, revived and rationalized by Plummer's convincing separation of exophthalmic goiter and toxic adenoma groups, and the use of x-ray in goiter treatment.

CLASSIFICATION (WITH SYNONYMS)

Goiters may be conveniently divided as follows (modified from Plummer):

1. Diffuse colloid goiter (simple-adolescent-colloid hypertrophy).
2. Adenomatous goiter, without hyperthyroidism (non-toxic adenoma).
3. Adenomatous goiter, with hyperthyroidism (toxic adenoma).
4. Exophthalmic goiter (Graves' or Basedow's disease), (hypertrophic parenchymatous thyroid).
5. Mixed type of hyperthyroidism (a combination of toxic adenomata and exophthalmic goiter).
6. Myxedema.
7. Cretinism.
8. Thyroiditis.
9. Malignant disease.

By far the most common types encountered are the first five of these diseases. We shall confine our remarks to them. It should be emphasized at the onset that the most important point in the clinical classification of goiter revolves about Plummer's well-established differentiation between true exophthalmic goiter and the toxic adenomatous goiter.

DIAGNOSIS

Goiter diagnosis is one of the most, if not the most, difficult diagnostic problem which confronts the internist and surgeon today, especially in the non-goitrous districts. It is really only a very careful study of disease for years that enables one properly to diagnose and treat these cases. So it is that without that experience a properly controlled and operated basal metabolism rate station is the most valuable single factor in recognition and management of these diseases.

A. *Simple Colloid or Adolescent Goiter*—This type of goiter is most commonly seen in young people between the ages of 12 and 25. The thyroid enlargement is uniform, smooth thyroid-shaped and without palpable nodules. These goiters do not show signs of toxicity, the basal

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metabolic rate being normal. They become bothersome from pressure, if large, and are in some cases quite disfiguring. They are considered physiologic in a way and tend to disappear spontaneously.

B. *Adenomatous Goiter Without Hyperthyroidism (Non-toxic Adenoma)*—In adenomatous goiters the thyroid presents an irregular, nodular enlargement. They are usually soft, though calcium deposit in a cystic adenoma may feel stony-hard like cancer, or an associated thyroiditis or hemorrhage may make the gland hard in consistency. The patients are usually 35 to 45 years of age, having had enlarged thyroids on the average for sixteen years before presenting themselves for treatment. The adenomata sometimes become very large with a tendency to interfere with respiration by pressure upon the recurrent laryngeal nerve and upon the trachea. They are sometimes substernal. Neither the vascular nor the nervous symptoms of hyperthyroidism is noted; the basal metabolic rate is normal. Their greatest danger is their tendency to become toxic, and if given iodin they will do so.

C. *Adenomatous Goiter with Hyperthyroidism (Toxic Adenoma)*—Toxic adenoma patients present themselves during middle life with a history of having had goiter for many years, but for the last few months an increasing weakness. The heart has become troublesome, the pulse rate rapid and perhaps irregular, and there may be shortness of breath on exertion and swelling of the feet. There is considerable nervousness and tremor, though these symptoms are more prominent as a rule in the exophthalmic type of goiter. It is most important to recognize that there has been a slow, steady development of these symptoms without remission. The basal metabolic rate is high, though not as a rule as high as in a "well-trained exophthalmic goiter case." The gland feels hard, but thyroiditis and carcinoma are likewise hard. The blood pressure varies from 160 to 180 systolic and from 80 to 90 diastolic with a pulse rate at rest of about 90 to 110. In the cases with cardiac decompensation, however, the rate may be very rapid and irregular from auricular fibrillation. We have a pulse pressure greatly increased, while in essential hypertension the diastolic blood pressure is elevated also, say with a reading of 180/110. There is a tendency to hypertension not seen in exophthalmic goiter (Plummer [10]). There is loss of weight although the patient has a good appetite. A "neuro" has a poor appetite, capricious, and does not lose weight. Excessive normal thyroxin output increases the basal metabolic rate. These toxic adenomata are nodular and, although hard, not so vascular as the exophthalmic goiter and, therefore, less often do we hear a bruit. Exophthalmos is never present in a purely adenomatous type of thyroid. Auricular fibrillation, dyspnea, and edema, may be present. About one in four cases usually shows auricular fibrillation, which disappears in most cases about two to three weeks after the operation. Some of these toxic adenomata produce serious vital organ degeneration without showing nervous symptoms. Colloid or non-toxic adenomata in neurasthenic people are often mistaken for hyperfunctioning goiters.

D. *Exophthalmic Goiter (Graves' or Basedow's Disease, Hypertrophic Parenchymatous Thyroid, Exophthalmic Goiter of Plummer)*—In exophthalmic goiter there is a sudden onset with a fluctuating course. The patient may be any age, but usually between 20 and 40. The disease runs in families and these patients are often optimistic, like tuberculosis subjects. There is a history of recent enlargement of the neck and a train of characteristic nervous symptoms, the latter often making their appearance before the former. The goiter is thyroid shaped, symmetrically enlarged, vascular, and smooth, unless adeno-

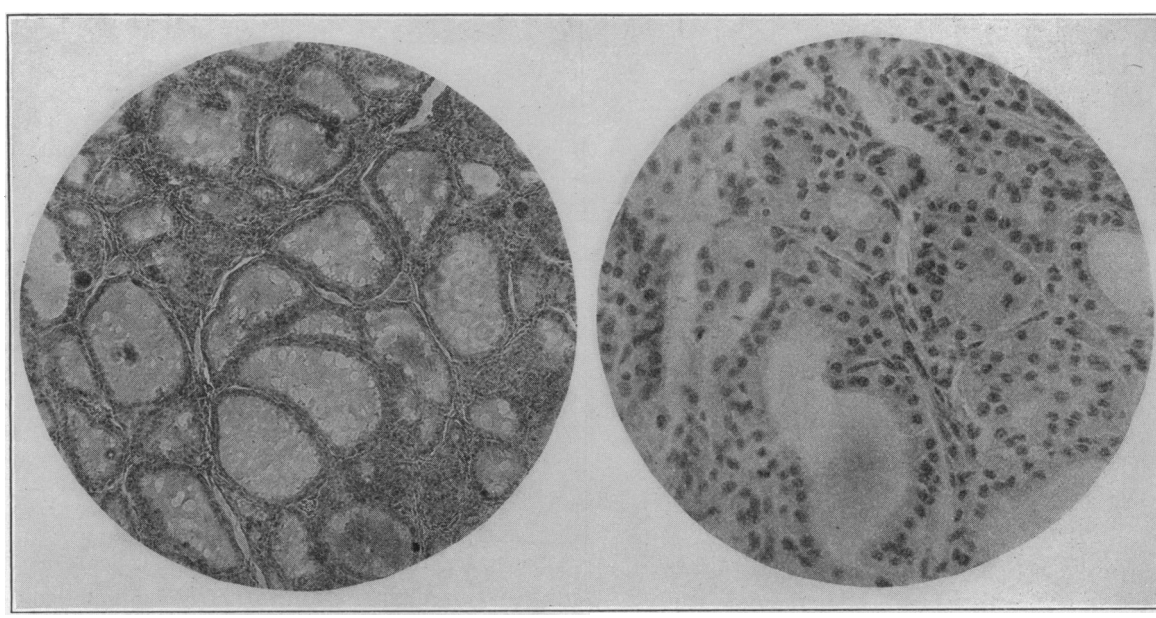

Fig. 1—Simple colloidal goiter.

Fig. 2—Parenchymatous hypertrophy. Solid area with columnar cells.

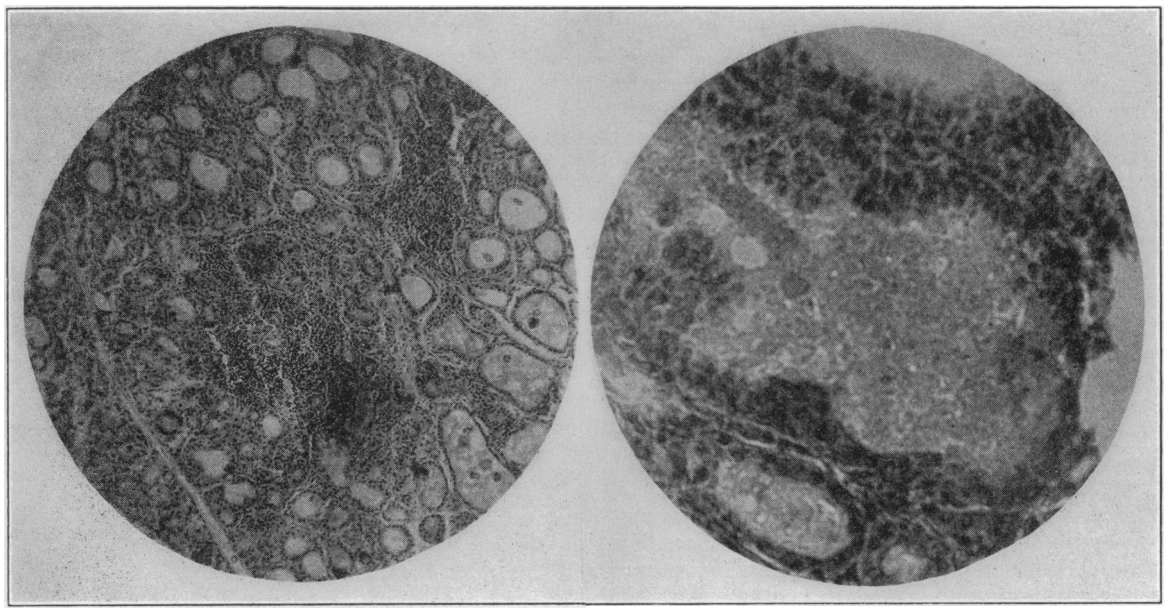

Fig. 3—Associated thyroiditis.

Fig. 4—Epithelia; cells desquamated into acini and colloid replacement in a hyperplastic area. Advanced stage of desquamation of cells and colloid replacement.

mata are present. The gland feels quite hard in the toxic cases. A bruit is often present; in 90 per cent of three-year cases and 80 per cent of three-month cases.[11]

The patient presents marked symptoms of nervousness and heat intolerance; less bed clothes are needed and also a lower room temperature. Tremor, tachycardia, perspiration, and muscle weakness are present. Exophthalmos is noted in the vast majority of cases, though true exophthalmic goiters can be present with a negligible degree of protrusion of the eyeballs. A large appetite with a concomitant loss in weight is characteristic—the "neuro's" appetite is variable and there is seldom marked weight loss. In young people the blood pressure may be 140/60. In patients over 40 it is usually 160/80 or higher. Note the high pulse pressure, the difference between systolic and diastolic. There is no tendency to hypertension. There may or may not be fever. The fingernails tend to separate from the matrix for an abnormal distance. Characteristic crises occur in which there is great weakness, nervousness even to dementia and associated nausea, vomiting, and diarrhea. Such crises occur much less frequently in toxic adenomata, but are often seen when there is an associated parenchymatous hypertrophy, extra-adenomatous. Some cases show jaundice from toxic hepatitis. Basal metabolic rates are useful in checking one's clinical judgment of the patient's condition. With an accurately controlled machine, the basal metabolic rate is of the utmost help in separating the nervous cardiac patient and the neurasthenic type from the true hyperthyroidism patient.

E. *Mixed Type of Hyperthyroidism* — The greatest difficulty in classification, diagnosis, and treatment lies in the mixed type of goiter with adenomata present in an exophthalmic goiter or in adenomatous goiter with an associated parenchymatous hypertrophy as in exophthalmic goiter. It must be because of the frequency of this type of disease that conflicting reports are heard regarding the efficacy or harm of Lugol's solution and why it is being recommended more and more for both types—exophthalmic goiter and toxic adenomata cases. This also may explain why prolonged use of Lugol's solution even in what is thought to be clear-cut exophthalmic goiter, does not permanently cure and really does harm, for with adenomata present, buried beyond recognition, we see iodin accentuating hyperthyroidism after the first preliminary short benefit. The symptoms and signs of these cases are a mixture of the toxic adenoma and the exophthalmic goiter patient with either one or the other predominating.

PATHOLOGY

Before discussing the treatment of the different types of goiter it is well to review their pathology, for here again the tissue changes underlying the different clinical pictures lend a guiding hand in treatment as well as prognosis, especially if the clinical diagnosis has been in doubt.

It should be emphasized at the start, however, that one cannot always tell from the examination of the tissue just what clinical state of toxicity existed in the patient at the time of thyroidectomy. This is particularly true of the adenomatous type of goiter with hyperthyroidism. Then again the histologic picture of the gland in cases receiving iodin for several weeks often shows involution changes, so marked that the diagnosis of exophthalmic goiter or of parenchymatous hypertrophy in an adenomatous goiter from tissue alone is difficult. A pathological classification, however, is useful and will definitely separate the adenomatous and the exophthalmic goiter disease groups. Let us look at them first from the gross, and then, the microscopic standpoint. In the gross alone there really are but three types of glands.

First, the smooth, thyroid-shaped or so-called butterfly goiter seen in simple colloid or adolescent cases and in the true exophthalmic or Graves' disease. Second, the nodular, irregular thyroid of the adenomatous group, toxic or non-toxic. Third, the mixed type, which, however, cannot be told in the gross before section. On section, in the gross, the cut surface in the colloid goiter is, as the name implies, glairy, sticky, and jelly-like, gray in color and with varying amounts of stroma and colloid. The exophthalmic type has a meaty appearance with little or no colloid escaping from the cut surface, unless Lugol's solution has been given for a few weeks. The color is that of partly cooked beef. The nodular, irregular, lumpy adenomatous type of goiter on gross section presents a varied picture, depending upon the admixture of varying amounts of colloid material, stroma, and the product of hemorrhage with its cyst formations and degeneration. Large collections of colloid substance surrounded by fibrous tissue envelopes are characteristic, and it is the hemorrhage into these that gives varying degrees of degeneration with ultimate fibrosis, hyalinization, and even calcification. The mixed type contains, as the name implies, varying amounts of adenomata and solid homogeneous tissue.

Microscopically there are five fairly distinct types of goiter, which, however, are usually confusing because of a tendency for two types to be present in one gland.

1. Simple, colloid or adolescent goiters show the enlargement due to an increased amount of colloid stuffing the acini and flattening out the epithelial cells. There is little cuboidal and no columnar epithelium. The gland is not vascular.

2. Exophthalmic goiter (Graves' disease) shows microscopically a marked hypertrophy of the epithelial cells lining the acini, changing the normal cuboidal or flat cell into a tall columnar affair. It is well to distinguish between hypertrophy as used here applied to epithelial cells, from the so-called hypertrophy of colloid material in simple goiter. There is also a multiplication of the number of cells or a true hyperplasia. The acini are small and their lumina contain little or no colloid material. There are, however, numerous fields of hypertrophied parenchymatous cells projecting into the cavity of the acini. The lymphatic and blood vessels are distended. Lymphocytic infiltration is a feature in most cases and it is thought indicative of a certain amount of thyroiditis. An estimation of the amount of thyroiditis may be helpful in prognosis, as it has often been noted that the gland with an excessive associated thyroiditis is the one about to go on to hypothyroidism postoperatively. The involution changes mentioned above after Lugol's solution have been well described among others by Rienoff [12] and Giordano,[13] and are as follows: Colloid material dilates the acini, producing a cystic condition so that the epithelial lining of the acini changes from a tall, columnar to a cuboidal or even flat type. The infoldings in part disappear and masses of cast-off degenerating epithelial cells may be seen in the acini. There is a relative increase in fibrous tissue. However, there are usually present some areas of the gland which show true parenchymatous hypertrophy from which a diagnosis may be made.

3. Adenomatous goiter, non-toxic. Microscopically the non-toxic adenomata show many areas little different from a normal thyroid and in other areas flattening and even absence of the epithelium lining the colloid or hemorrhage-stuffed acini. Masses of undifferentiated fetal cells abound in almost all adenomatous goiters.

4. Adenomatous goiter, toxic. This gland differs from the non-toxic in one respect only and that is that it contains certain areas of hypertrophy of the parenchymatous cells lining the acini with a change from flat or cuboidal to a columnar type of cell and with infoldings of these enlarged cells into acini even as is seen in exophthalmic goiters.

This is the type of goiter in which the pathologic tissue picture is less apt to mirror accurately the clinical condition of the patient. Although the microscopic picture of the gland at times shows associated parenchymatous hypertrophy in a degree comparable to the toxic state of the patient, it does not always give one an accurate estimation of the state of clinical toxicity.

5. Mixed type. The mixed type of gland shows either a predominantly adenomatous type with an associated parenchymatous hypertrophy either intra- or extra-adenomatous or a true exophthalmic goiter type with adenomata buried in its substance. The mixed type of gland with adenomata buried in hypertrophic parenchymatous tissue or the exophthalmic with associated adenomata is often correctly diagnosed by the pathologist, but the clinical state of toxicity is here again difficult to predict. The giving of Lugol's solution for long periods preoperatively makes the pathologic differentiation of the excised glands more difficult than formerly.

To sum up the pathology, then: The simple colloid and the exophthalmic goiter glands are accurately recognized. The nodular adenomatous goiter, be they toxic or non-toxic, fall into one group. In the gross, differentiation between these two is not possible, and in the microscopic study, though in a fair percentage of cases a pathologist can pick out the toxic cases, the general average of correct diagnoses is not sufficiently high for practical purposes.

PREVENTION

Before taking up the treatment of goiter it is well to say a few words about its prevention. This is, of course, accomplished insofar as our present knowledge permits in the judicious use of iodin. Remember that the cause of goiter is as yet not known. McCarrison [14] has shown through feeding experiments that iodin deficiency is the chief known factor involved in goiter production. He states that it is either a breakdown in assimilation in the intestinal canal and cites fecal bacterial contamination in unhygienic conditions as a cause of iodin deficiency or a breakdown in utilization of iodin by thyroid or body cells after absorption. The lack of iodin plus the

unknown "X" then gives a colloid goiter. Iodin is given to prevent this. If colloid is already laid down, thyroid extract enough to bring metabolism to normal is given to dissolve the colloid.

Iodin is given as prophylaxis hesitatingly in some instances, for fear of causing a normal gland to "hyperfunctionate." No amount of iodin will do this. If harm is done, you may be sure small unrecognized adenomata were present to be whipped into action. It should never be prescribed prophylactically in the presence of adenomata. Marine and Kimball [15] have shown in school children in Akron, Ohio, that the danger of giving iodin to children in this country is negligible, even if a small colloid goiter is present. This holds up to the age of 20. The older the patient and the larger the goiter the more likely is the gland to contain adenomatous tissue, although palpation may fail to reveal its presence, and the more danger in giving iodin.

Iodin administration affecting an entire community such as iodized salt or an iodized water supply seems quite inadvisable because of the certainty of producing hyperthyroidism in quiescent adenomata present in a colloid or otherwise normal thyroid gland. Prophylactic measures should apply particularly to children and consist in giving iodin in the form of sodium or potassium compound in the amount of one-tenth grain once a week between the ages of 11 to 17, as recommended by Marine. It is important to give childbearing women of goiter families prophylactic iodin also, for this prevents goiter both in the mother and the child.

To repeat, then, iodin as a preventive measure against goiter, is absolutely safe for children who do not have goiter. It is quite safe in children with small colloid goiters up to the age of 20. For patients over 20, for young patients with large goiters, or for those in whom nodules can be felt, it should not be given or hyperthyroidism might result.

TREATMENT

Let us emphasize again that the proper treatment of thyroid disease depends upon the recognition of the type of goiter in question. The different types are treated as follows:

1. *Simple Goiter (Colloid)*—Treatment is medical unless adenomata or toxicity develop, and consists in the administration of small doses of iodin to the small and early cases and desiccated thyroid to the larger ones of long standing. Iodin alone will not always produce absorption of colloid already laid down. In giving thyroid extract symptoms of increased basal metabolism must be carefully watched for and, if practicable, basal metabolic rate estimations done from time to time. Remember that the effect of desiccated thyroid or thyroxin is cumulative—the one day's dose often reaching its maximum effect a week later, and taking two to three weeks to entirely pass off. Nervousness, tremor, palpitation, and tachycardia are early symptoms of overdose and if the drug is continued, headache, pains, and aches in the muscles and bones with loss of appetite and nausea follow.

2. *Adenomatous Goiter Without Hyperthyroidism*—It is safe to say that one in four,[16] 25 per cent, of this type of goiter becomes toxic after the age of 35. They should therefore be considered surgical if the adenomata reach three-fourths of an inch or more in diameter and if the patient is over 30 years of age. It is because adenomatous goiters tend to recur, if removed at an early age, that we delay operation to age 25 at least. Disfigurement and pressure symptoms also call for operation. Iodin should never be given.

3. *Adenomatous Goiter with Hyperthyroidism*—It is because the symptoms of hyperthyroidism in these cases are due to the fact that the goiter has begun to throw out into the circulation an increased amount of normal thyroxin that iodin is not required. Lugol's solution is given, however, for a few days previous to operation for fear of there being unrecognized, associated parenchymatous hypertrophy which might throw the patient into a postoperative crisis, if iodin were not given. All cases are considered surgical, if patient is over 25 years of age, which procedure is curative provided the adenomata are removed before vital organ degeneration has progressed to any extent. Recurrence is rare if the operation has removed all adenomata. Cardiac symptoms predominate, but often clear up on rest in bed alone without giving digitalis. If the cardiac decompensation and auricular fibrillation persist, a digitalis compound may be given. However, it is well to discontinue it for a few days before operation. It seems fair to suppose that quinidin may be substituted for digitalis at this stage.

4. *Exophthalmic Goiter (Graves' Disease)*—Operation is indicated if the patient is seen before or after a crisis, but never during one. Rest in bed and Lugol's solution are usually all that is necessary to prepare most patients. Basal metabolic rates are necessary in checking one's clinical judgment of the patient's condition. All cases should be operated upon after appropriate preoperative treatment, if aged 25 or over. If another surgical condition is present, thyroidectomy should be done first unless it is an emergency, such as ruptured appendix, duodenal ulcer, or some such catastrophe.

There is about a five per cent chance of recurrence which might make a second operation necessary or the use of x-ray treatment advisable. It has not yet been demonstrated that iodin actually cures exophthalmic goiters. Doctor Boothby [17] says: "The drug improves and apparently holds in abeyance the nervous, mental, and gastro-intestinal symptoms which are so characteristic of exophthalmic goiter. It also prevents the development of the fatal postoperative exophthalmic goiter crisis. The metabolism is remarkably decreased in about one-third of the cases, somewhat less markedly affected in another one-third, while in the remainder, no demonstrable effect in the metabolism is produced. In general, therefore, according to the evidence available at the present time, iodin should be advised with few excep-

tions, only as a temporary therapeutic measure, in the treatment of exophthalmic goiter to bring the patient into a safe condition for a partial thyroidectomy."

5. *Mixed Type*—According to our classification this is a toxic goiter and should be treated surgically after appropriate preoperative preparation. Lugol's solution is indicated, for as the name implies, part of the hyperthyroidism is due to iodin-deficient secretion of the hypertrophic parenchymatous part of the gland. Iodin does not cure.

PREOPERATIVE TREATMENT

The principles of this treatment consist in restoring body fluids, replacing reserve glycogen and body weight, and increasing hemoglobin and reducing nervous tension.

This is accomplished by giving a high caloric diet, 3000 to 5000 calories, dependent somewhat upon appetite, forcing fluids, saline and glucose solutions by subcutaneous proctoclysis, or intravenous routes, and occasionally by giving transfusions. Crile [18] has shown that transfusions of whole blood not only improve hemoglobin and increase body fluid, but have a very beneficial effect on a goiter toxic patient's nervous syndrome. I have found that the administration of salt solution intravenously seems to benefit very sick patients more quickly if given slowly and repeatedly in large amounts than when given by the subcutaneous method. Glucose solutions should also be administered this way. Sedatives are given freely. Patient should rest but not be made bedridden, being allowed up as much as possible.

LUGOL'S SOLUTION

The exophthalmic goiter is the type par excellence which requires iodin in its treatment, for, as Kendall [19] has shown, the thyroid gland is throwing into the circulation an excessive amount of abnormal thyroxin whose molecule is deficient in iodin. The exophthalmic goiter patient needs iodin. Iodin is administered preferably in the form of Lugol's solution, which is the U. S. P. Liquor iodi compositus. It contains 5 grams of iodin and 10 grams of potassium iodid in 100 cc. of water. In each cc. of the solution there are 126 mgm. of iodin; in 10 cc. 78 mgm. or 1.2 grains. If the patient vomits Lugol's solution, repeat the dose immediately and repeatedly until it is retained. Patients soon tolerate it. It may be given in subcutaneous fluids, by rectum, or even by duodenal tube. It is best given in the average case in grape juice, and in amounts of ten to forty drops three times a day, depending upon the toxicity of the patient. Too much is rarely, if ever, given and often a far too small amount is tried. Its beneficial effect starts in from two to four hours and lasts eight to ten days, stopping the extreme nervousness and vomiting following crisis. However, the basal metabolic rate does not start down until from eight to fourteen days. Lugol's solution is given for a short period preoperatively to exophthalmic goiter patients, and postoperatively for two or three months in the very toxic cases. It may be used as a diagnostic test, for if given for two weeks preoperatively with failure to reduce the pulse rate and the basal metabolic rate, as it usually will in a pure exophthalmic case, you are dealing with a toxic adenomata or a mixed type of case. It is well to give a large dose of Lugol's solution per rectum for several doses in a case of exophthalmic goiter in crisis with vomiting. No discussion of the use of Lugol's solution is complete without emphasis of the note of warning resounding from all sides that the indiscriminate use of iodin for long periods of time in almost any type of goiter is doing untold harm to many patients. The lay public is grasping at Lugol's solution as a panacea for all goiter disease. Doctors should prevent this.

POSTOPERATIVE TREATMENT

This consists in a continuation of medical regimen as outlined preoperatively with addition of more sedatives and for a time increased amounts of Lugol's solution. Morphin sulphate may be used freely. Special alertness must be exercised by the attending physician to recognize and appropriately handle the many postoperative complications which may arise, such as infection, tracheitis, laryngitis, tetany, and cardiac decompensation, and especially a rapidly developing bilateral hydrothorax. The avoidance of operative and postoperative deaths and complications depends upon a painstaking and conscientious cooperation between the surgical, medical, and laboratory services. Such teamwork is essential to the proper handling of toxic goiter patients. This is seldom available outside of the large hospital or clinic.

OPERATIVE INDICATIONS

The indications for operation are:

1. Increasing quadriceps power.
2. Gaining weight.
3. Lessened nervous instability.

It is best if the basal metabolic rate is reducing, but this is a variable factor. Always operate upon the thyroid first if disease is present in some other organ also. Secondary infections in toxic goiters subside on Lugol's solution. It is well to double the dose of Lugol's solution the day preceding, during, and after an operation.

X-RAY TREATMENT

At present the x-ray treatment of goiter is where its surgical treatment stood twenty years ago. Sufficient statistical material on treated cases is not yet available for a just comparison with those treated by surgery. Until x-ray has proven of more definite value it seems fair to state that it should be used in cases under twenty years of age, very early cases, and in a selected group of cases where for some reason operation seems inadvisable, and postoperatively where further destruction of gland tissue is needed.

It should be borne in mind that even surgical treatment is but one of the many steps in the successful treatment of a goiter-bearing patient, which if undertaken alone without pre- and post-

operative medical and laboratory supervision, will often result disastrously.

SUMMARY

1. The most important point about thyroid disease is recognizing goiter early before vital organ degeneration has taken place.

2. Next we must accurately differentiate the type of goiter and the degree of hyperthyroidism present in order to give the proper treatment.

3. The commoner types of goiter are:

(a) Colloid goiter (simple colloid hypertrophy, adolescent, diffuse colloid).

(b) Adenomatous goiter without hyperthyroidism (non-toxic adenoma).

(c) Adenomatous goiter with hyperthyroidism (toxic adenoma).

(d) Exophthalmic goiter (Graves' or Basedow's disease (hypertrophic parenchymatous thyroid).

(e) Mixed goiter with hyperthyroidism.

4. Pathologic tissue examination divides the commoner types of thyroid glands into simple colloid goiter, exophthalmic goiter, adenomatous goiter and a combination of the last two, but fails to indicate accurately the clinical state of the patient in adenomatous goiter; *i. e.*, toxic or non-toxic, in a sufficiently high percentage of cases for practical use.

5. The administration of Lugol's solution produces a cystic and colloid replacement of the hyperplastic and hypertrophic parenchymatous cells in both toxic adenomata and true exophthalmic goiter glands.

6. Prevention of goiter by iodin administration is absolutely safe for school children, but dangerous for older people who may have adenomata in their thyroids. Therefore it should be given individually and not wholesale as in iodized salt or water.

7. Lugol's solution does not in itself cure exophthalmic goiter, being merely the most important adjunct to surgery or x-ray treatment. It should never be given to patients having non-toxic adenomatous goiters, but should be given to those who suffer from the adenomatous goiters with hyperthyroidism, and to patients with the mixed types, for a few days preoperatively for fear of an associated parenchymatous hyperplasia and hypertrophy.

8. A careful and conscientious cooperation between the surgical, medical and laboratory services is essential to the proper handling of thyroid disease, and such teamwork is seldom available outside of the large hospital or clinic.

9. A properly conducted and controlled basal metabolic rate machine is essential to the proper handling of disease of the thyroid unless one has had years of experience with such patients.

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UNILATERAL SIGHTING*

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DISCUSSION *by Roderic O'Connor, M. D., San Francisco; Frederick C. Cordes, M. D., San Francisco; P. Obarrio, M. D., San Francisco.*

A REVISION of the current explanations of the physiological and optical phenomena of binocular vision is resulting from the recent work of Parson [1] and from that of the author,[2] on eyedness and handedness.

Sheard [3] says, in discussing this work editorially, "Our clinical tests upon the elements of convergence, fusion powers and muscular insufficiencies are based upon the principle of triangulation, in which the line joining the nodal points of the two eyes—or interpupillary distance—serves as the base of the triangle, while the point of fixation is made to lie on a line drawn as perpendicular bisector to the base line so that the distances from the nodal point of each eye respectively to any point on the median line are equal. Binocular single vision is thus graphically diagrammed and discussed as though each of the two eyes was equally dominating and directing

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